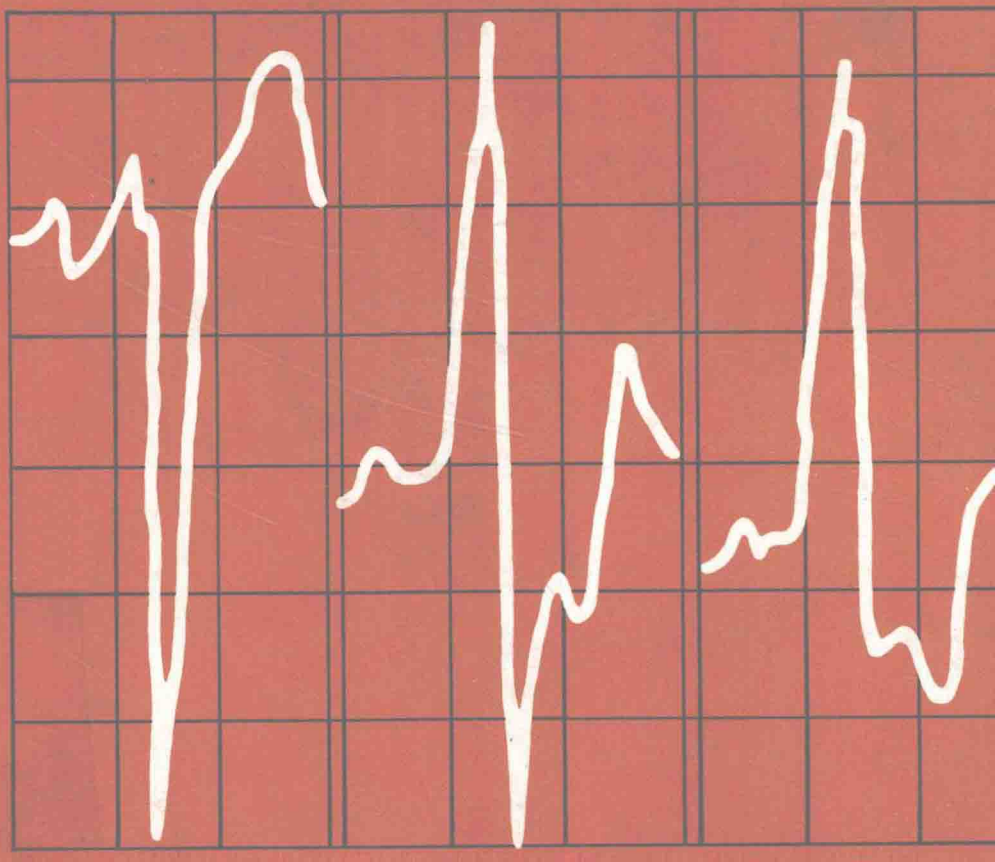
# An Introduction to Electrocardiography

*Second Edition*

John Hamer



# An Introduction to Electrocardiography

A Primer for Students, Graduates, Practitioners and Nurses concerned with Coronary Care and other forms of Intensive Care

*Second Edition*

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# Preface

The increasing demands of the medical curriculum make it difficult to find a place for electrocardiography. A basic knowledge is required for qualifying examinations, and the pre-registration house officer often finds himself in urgent need of a certain degree of practical skill. The greater sophistication of family doctoring in recent years also makes many practitioners eager to acquire some ability in electrocardiographic interpretation. The increasing involvement of nurses in E.C.G. monitoring for coronary care and other forms of intensive care has produced a new group needing to acquire the basic skills of E.C.G. interpretation. This book aims to provide a simple introduction to electrocardiography for these purposes, and to provide a foundation for more detailed future study if required.

The main clinical value of the electrocardiogram is in the interpretation of arrhythmias, in the diagnosis and management of ischaemic heart disease, and in the assessment of ventricular hypertrophy. In addition, a number of abnormalities of conduction such as bundle branch block, which cannot easily be diagnosed by other methods, are revealed by the electrocardiogram. The interpretation of the electrocardiogram has necessarily developed on an empirical basis, and there has been a tendency in the past to consider an 'electrocardiographic diagnosis' unrelated to the clinical situation. Changes suggesting ischaemic heart disease are particularly liable to be treated in this way as the symptoms may be difficult to evaluate and the physical signs unimpressive. The

present work aims to avoid these pitfalls and to provide a pragmatic approach to the subject closely linked to clinical work. Our understanding of the electrical changes in the myocardium is now approaching the level at which a logical interpretation of the electrocardiogram can replace an empirical one. The graduation of electrocardiography from an art to a science makes it an easier subject for the beginner, as deduction can replace learning on the basis of pattern recognition alone. It is hoped that this approach will give the reader a useful addition to his or her clinical tools with relative ease.

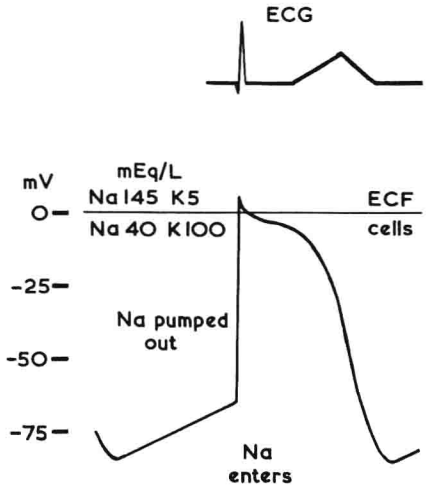
# Contents

# 1

# Physiological Basis of the Electrocardiogram

## Source of the Electrical Activity Recorded

The electrocardiogram is a record of voltage changes at the body surface produced by alterations in the intracellular potential of the myocardial cells. The electrical potential within the cell is related to the difference between the concentrations of intracellular and extracellular electrolytes (1). The main cation of the extracellular fluid is sodium, and of the intracellular fluid, potassium. The intracellular potential is negative to that in the surrounding medium, i.e. the cell membrane is 'polarised' until the cell is stimulated, when there is a brief reversal of electrical potential (depolarisation) associated with a rapid influx of sodium ions. Some specialised cells, for instance, in the atrioventricular (AV) node, rely for activation on a more gradual influx of calcium ions, giving a slower rise in electrical potential and associated with slower conduction from cell to cell; this calcium current contributes a minor component to the late stage of activation of all myocardial cells. The potential difference across the cell membrane remains close to zero for most of systole and the resting situation is restored (repolarisation) by a more gradual efflux of potassium ions. A metabolic process in the cell membrane (the sodium pump) maintains the electrolyte balance by extracting sodium in exchange for potassium ions against the concentration gradient. The initiation of contraction of the myocardial cell is associated with movement of calcium ions into the region of the




## 1. Membrane Potential

The negative potential within the cell at rest is related to the properties of the cell membrane, that is, to the high potassium and low sodium concentrations within the cell relative to the extracellular fluid (ECF). When the cell is activated the cell membrane ceases to be a barrier to the entry of sodium ions into the cell. This disturbance of the intracellular environment, or a similar movement of calcium ions, produces contraction of the myofibrils leading to systole. The resting membrane potential is restored (repolarisation) by a more gradual outwards movement of potassium ions. An active metabolic process in the cell membrane, the 'sodium pump', extrudes sodium ions in exchange for potassium ions and maintains the difference in ionic composition between the cells and ECF.

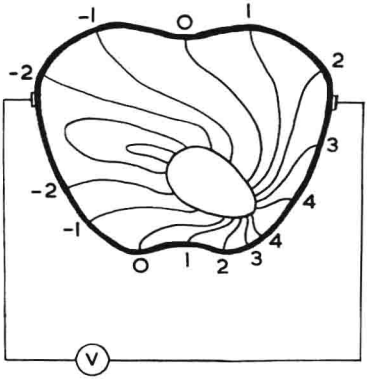
As in this diagram, cells capable of spontaneous rhythmical activity show a gradual rise in resting potential throughout diastole until a threshold is reached, which triggers the next cycle. Other cells are triggered by the activation of adjacent cells.

The electrocardiogram is the summated effect of the electrical changes in all the myocardial cells. The QRS complex represents the rapid rise of the intracellular potential in the ventricular muscle, and the T wave the slower corresponding recovery process.

myofibrils, either by entry through the cell membrane or as a secondary effect of the entry of sodium or calcium ions. The electrical changes at the cell membrane that give rise to the electrocardiogram are related only indirectly to the subsequent myocardial contraction. Although changes in the electrocardiogram occur in myocardial hypertrophy, it is not possible to make any deductions from these changes with regard to the quality of myocardial performance.

## Behaviour of the Heart as a Single Electrical Source

These changes in intracellular potential produce electrical currents in the surrounding tissue which spread to the surface of the body, where they produce changes in electrical potential (2) which are




**2. Electrical Field of the Heart**

The changes in the myocardial cells with each cardiac cycle produce a varying electrical field within the body. The consequent potentials at the surface of the body are recorded as the electrocardiogram. A hypothetical field (in mV) at one instant in the cardiac cycle is shown in the diagram. A bipolar lead, as shown in the diagram, measures the difference in potential between two points on the body surface.

recorded as the electrocardiogram. The electrocardiogram is therefore an indirect measure of cardiac electrical activity. A recording at any point on the surface of the body is influenced by changes in all parts of the heart. The effects of parts of the

heart closest to the recording electrode are much less than would be expected from the distance involved, as the heart is largely separated from the surface by tissue of poor electrical conductivity such as fat and air-filled lung. The electrical disturbances produced by the electrical discontinuity at the surface of the body, and by the highly-conductive blood in the heart, accentuate this effect, so that for most purposes the heart can be regarded as a single localised source of electrical activity near the centre of the thorax.

## SEQUENCE OF ELECTRICAL EVENTS IN THE HEART

### Production of the Pattern of the Electrocardiogram for each Heart Beat

The whole sequence of activation and recovery of the myocardium in each cardiac cycle gives rise to a pattern of electrical events at the body surface. The recording system of the electrocardiograph, with exploring (positive) and reference (negative) electrodes (2), is arranged so that as the process of activation moves towards an exploring electrode, an upright (positive) deflection is produced. The sequential spread of activation and recovery through the heart, depending on the anatomical arrangement of the atria and ventricles and the specialised conducting tissue, produces a series of deflections in the electrocardiogram which are labelled arbitrarily from the middle of the alphabet, P, Q, R, S, T and U. For practical purposes the recording electrode may be regarded as on the left or right side of the heart and the changes in these two electrodes are generally in opposite directions. The pattern of spread of the activation process allows us to interpret these deflections in terms of electrical changes in different parts of the heart.

### Function of the Specialised Conducting Tissue

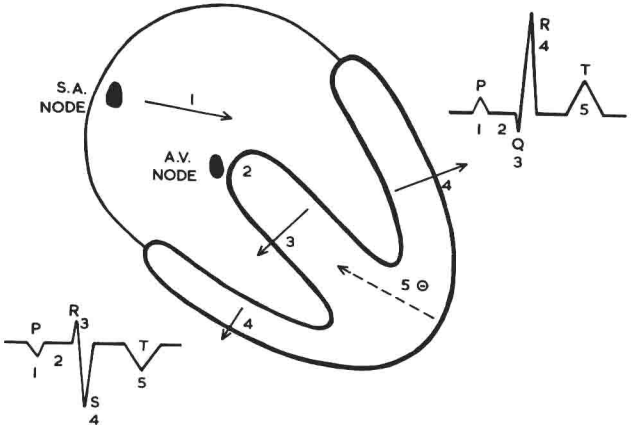
Modified myocardial cells having spontaneous rhythmical activity and being capable of the initiation of activation of the whole process of cardiac contraction are found in the sino-atrial node, in

atrial muscle, the atrioventricular junction and in the bundle of His and its branches. The other myocardial cells have lost this property in the process of differentiation into primarily contractile apparatus. Some of the modified cells, in the bundle of His and its branches (Purkinje tissue), are also specialised for the rapid transmission of the impulse of progressive activation from one cell to the other, which proceeds in this tissue at about three times the speed found in unspecialised myocardium. Cardiac contraction is initiated by the part of the heart that has the fastest natural spontaneous rate. This is normally the sino-atrial node, and activation here spreads to the adjacent tissue, and eventually to the whole heart, to trigger the cardiac cycle, suppressing other potential pacemakers with slower intrinsic rates.

The activation process arising in the sino-atrial node at the junction of the superior vena cava and the right atrium sweeps to the left, anteriorly and downwards across the atria, and produces a small upright deflection in a left-sided lead and a small negative or diphasic deflection in a right-sided lead (3). This deflection is labelled the P wave and represents the summated effect of the electrical activation of all the atrial muscle fibres. It seems likely that certain preferential pathways run between the sino-atrial and atrioventricular nodes as, occasionally under certain circumstances (*see* p. 77) normal conduction may appear to continue in the absence of P waves (sino-ventricular rhythm).

In the lower part of the atrial septum the process of activation spreads to the atrioventricular node which delays the entry of the impulse into the ventricle until atrial systole is completed. This delay, which is important to ensure proper ventricular filling under stress, is by convention described as the PR interval, measured from the beginning of the P wave to the first deflection of the QRS complex, whether this is Q or R (3), and although it varies with heart rate, being longer at slower rates, is normally less than 0.22 s. The activation process then spreads down the bundle of His and its branches to the ventricles.

The bundle of His consists of specialised conducting tissue which runs from the atrioventricular node through the atrioventricular ring into the upper part of the ventricular septum, where it divides into branches which ramify in the subendocardial region of both ventricles. The original description of an initial division to left and right bundle branches now seems to be an




**3. Formation of the Electrocardiogram**

The complex wave form of the electrocardiogram is produced by sequential activation and recovery of the atria and ventricles. The impulse arises in the sino-atrial node and passes through the atria in a predominantly leftward direction, producing the P wave in the electrocardiogram, 1, eventually to activate the atrio-ventricular node, 2, where there is delay for a fifth of a second; the bundle of His and its branches conduct the impulse to the main mass of the ventricular muscle fibres where activation begins in the septum from early branches from the bundle of His, 3, passing from left to right. Simultaneous activation of the free walls of the ventricles, from within outwards, produces the major deflections of the QRS complex, 4. The recovery process in the ventricles, 5, is in the opposite direction, but, as the electrical changes are in the reverse sense, the deflection in the electro-cardiogram (T) is in a similar direction to the main wave of the QRS complex.

oversimplification of the situation. It seems likely that the main trunk gives off a series of small branches to the left side of the septum, including those forming the posterior division of the left branch to the postero-inferior regions of the left ventricle, and then continues as an intact bundle for a short distance before

dividing to a right branch, which has a long course in the right subendocardial region of the interventricular septum before dividing further to form a subendocardial network throughout the right ventricle, and the anterior division of the left bundle branch which passes to the anterolateral region of the left ventricle where it also branches into a subendocardial network, the function previously attributed to a left bundle branch being carried out by the relatively separate anterior and posterior divisions. The cells of the bundle of His and its branches (Purkinje tissue) are specialised for the rapid conduction of the impulse, and the anatomical arrangements allow nearly simultaneous activation of all parts of the ventricles. The details of the branching of the bundle of His assume importance in the assessment of conducting system disease (*see* p. 118). The impulse spreads very rapidly in Purkinje tissue, and the number of fibres involved is small, so that no electrical charges are usually recorded at the surface of the body at this time. The His bundle potential can be detected using a bipolar intracardiac electrode placed across the tricuspid valve at cardiac catheterisation, a technique that has been very useful in the assessment of abnormalities of atrioventricular conduction (p. 115). It is only when the process reaches the terminal Purkinje fibres beneath the endocardium of the ventricles and the main mass of ventricular muscle begins to be activated, that sufficient voltage is produced to give deflections in the conventional surface electrocardiogram.

## Ventricular Septum

The first part of the ventricles to be activated is the ventricular septum, by early fibres of the left branch of the bundle of His which arise after a very short course in the septum (*see* p. 5). The main bulk of the septum is made of muscle that is embryologically and functionally part of the left ventricle. The process of activation therefore begins subendocardially on the left side of the septum and spreads towards the right (3). The process is spreading away from a left-sided electrode and so produces a downward deflection in left-sided leads, which is conventionally labelled the Q wave. The corresponding upward deflection in a

right-sided lead is, by convention, labelled R; the letter Q is used only for an initial downward deflection in the whole group of changes produced by ventricular activation (QRS complex).

## The Free Walls of the Ventricles

The deflection in the electrocardiogram from septal activation is quickly followed by activation of the main mass of the ventricular muscle. The process spreads outwards from the termination of the Purkinje fibres just beneath the endocardium in all parts of the ventricles, and the electrocardiogram records the summated effects of the resulting outward electrical forces. As the left ventricle is much bulkier than the right it normally dominates the electrical process, and potentials recorded by both left and right-sided electrodes seem to be entirely derived from the left ventricle (3). Right ventricular activation is not separately evident, even at a right-sided electrode, and its only influence is to cancel out some of the simultaneous electrical activity from the left ventricle. During activation of the main ventricular muscle mass, a left-sided electrode records a large upwards deflection (R); at the same time a right-sided electrode records a large downwards deflection (S). There is often a small further deflection in the opposite direction due to the late spread of the impulse to part of the base of the right ventricle. The whole process of ventricular activation is known as the QRS complex. The normal QRS pattern is altered when the activation process in the ventricle is disturbed, as in ventricular hypertrophy or cardiac infarction.

## Recovery Process (Repolarisation)

During the next stage of the cardiac cycle there is little difference in potential across the cell membrane and the electrocardiogram remains at the base line (ST segment). The recovery process begins during this interval and produces the final large and wide deflection, the T wave. The electrical events of recovery are opposite in sense to activation and produce changes in the reverse

direction in the electrocardiogram, i.e. a downwards deflection as the process moves towards the recording electrode. In general, recovery is a much slower process than activation. Atrial recovery is not usually evident because it is swamped by the QRS complex, but may lead to slight depression of the first part of the ST segment when the rate is fast, as on exercise. The summated effect of the recovery process from all parts of the ventricles produces a wide, prolonged deflection that is labelled T whether it is upwards or downwards in direction. The pathway of the recovery process in the ventricles is almost the reverse of activation, spreading inwards from the epicardial surface; nevertheless, as the underlying electrical changes in the heart are opposite in sense to those producing activation, the T wave is usually similar in direction to the main wave of the QRS complex, in spite of the reversal of direction of the process in the heart. An upright deflection is, therefore, usually found in left-sided leads and a downwards deflection in right-sided leads (3). The T wave usually rises more gradually than it falls, and symmetrical 'spiky' or 'peaked' T waves are often abnormal.

A small additional deflection, the *U wave,* probably also part of the recovery process and normally in the same direction as the T wave, is sometimes seen. The U wave may be inverted in leads with dominant R waves in left ventricular hypertrophy and in ischaemic heart disease. For simplicity it is generally removed from the illustrations here, apart from hypokalaemia (36, 43) where a prominent U wave is a major feature.

The recovery process, represented by the ST segment and the T wave, is much more variable than the QRS complex, and changes in this part of the electrocardiogram are frequent in response to autonomic disturbances, electrolyte imbalance, and minor myocardial ischaemia (primary T wave changes), and must necessarily follow disturbances of activation, to be described in later chapters, such as ventricular hypertrophy, BBB, WPW syndrome, ectopic beats and aberrant conduction (secondary T wave changes).

*Normally inverted T waves.* The normal T wave axis is similar to but slightly to the left of the QRS axis (p.12) and therefore right-sided leads such as $V_R$ and $V_1$ normally have inverted T waves.